

Paramedic Resource Manual

SHOCK SECTION FOUR

2005 Update by
Ontario Base Hospital Group Education Subcommittee

OBJECTIVES: SHOCK

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. define shock.
2. classify the types of shock.
3. briefly describe the pathophysiology and distinguishing clinical features of each type of shock.
4. for each type of shock, state prehospital situations in which the provider should anticipate the development of shock.

If you have studied this subject previously, you may test your ability using the self-assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.

INTRODUCTION

The approach to and management of the patient in shock requires a systematic, organized process based on the underlying pathophysiology and an understanding of the fundamental principles of resuscitation. This module will focus on:

1. A useful definition of shock.
2. A number of important points with respect to the diagnosis of shock.
3. A brief explanation of the pathophysiology at a cellular level.
4. A classification of the types of shock.
5. An overview of the patient's compensation to reduced tissue perfusion.
6. A grading system for the severity of hemorrhagic shock.
7. The principles of assessment and management in the prehospital setting.

DEFINITION

Shock is a clinical state in which there is a widespread reduction of **tissue perfusion** resulting in:

- o inadequate **oxygenation** at a cellular level
- o inadequate removal of toxic metabolic by-products which, if prolonged, leads to a generalized impairment of cellular function and ultimately cellular death.

The key words in defining shock are **perfusion** and **oxygenation**.

IMPORTANT POINTS

The words **hypotension** and **shock** are **not synonymous**. Many individuals will be encountered in the prehospital environment with blood pressures of 90/50, which is "normal" for that particular patient. This particular individual remains warm, well perfused, and well oxygenated at a cellular level. In contrast are patients with a blood pressure of 120/70, who may in fact be in shock. This individual may **normally** be markedly hypertensive, and now clinically is vasoconstricted, peripherally cyanosed, and poorly perfused. Therefore, not only are hypotension and shock not synonymous, but a **normal** blood pressure does not ensure adequate cellular oxygenation. As will be emphasized in this module, it is the **entire clinical picture** of the patient which determines the adequacy of tissue perfusion.

THE CELLULAR LEVEL

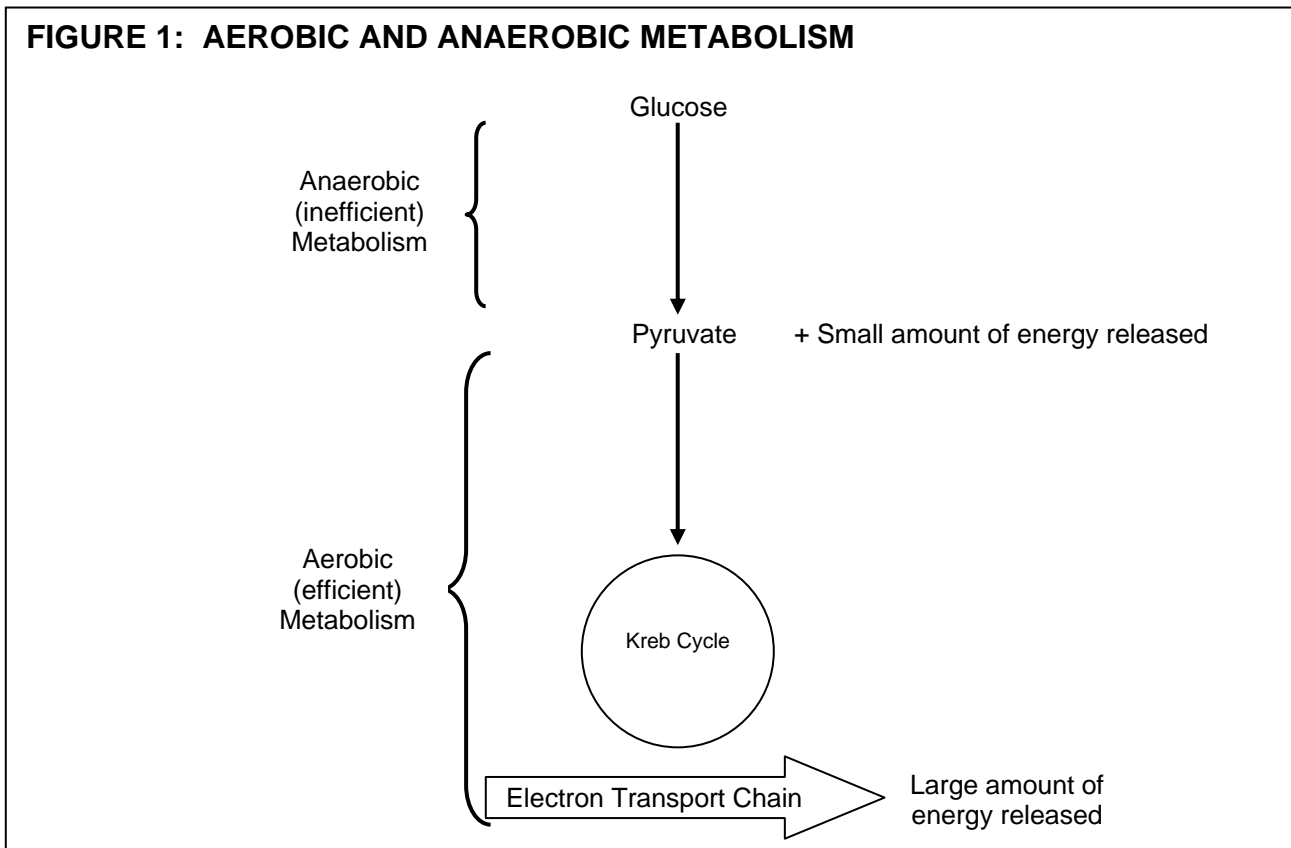
Prior to explaining the disruption which occurs at a cellular level, it is important to understand the principles of cellular metabolism. Cells (which in large numbers combine to make up tissues; tissues combine in large numbers to make up organs) can utilize three principal sources for the energy necessary for routine cellular functions, **i.e.** Carbohydrates, proteins, lipids. These sources are necessary for producing cellular constituents, maintaining a functioning cellular membrane, performing special functions depending on the specific organ.

CARBOHYDRATES

Under most circumstances, cells will preferentially utilize carbohydrates, in the form of glucose. When glucose is metabolized by cells, energy is stored in the form of adenosine triphosphate (ATP), an energy rich molecular compound.

Two processes are available for metabolism of glucose:

- o The first is inefficient, expensive for the cell and occurs in the absence of oxygen, **i.e.** an anaerobic environment. This process is known as **glycolysis** which produces 2-3 ATP and lactic acid as a by-product.
- o The second process is efficient and occurs in the presence of oxygen, **i.e.** an aerobic environment. This is known as the **Krebs Citric Acid Cycle** and runs in combination with the **Electron Transport Chain** producing 36-37 ATP and CO₂ as a by-product which is easy to eliminate.



Under most circumstances most cells in the body utilize the efficient pathways in an aerobic (oxygen rich) environment.

When cells suffer from a generalized decrease in perfusion, there is a reduction in the delivery of oxygen to cells. The rapidity with which this occurs and the host's ability to compensate (outlined below) will determine the ultimate outcome.

If the reduction in oxygen delivery to the cell persists, anaerobic or inefficient metabolism occurs. The process of glycolysis yields pyruvate as an end product which is subsequently metabolized to lactic acid. The accumulation of lactic acid results in a metabolic acidosis within and outside the cell.

Necessary cellular functions (determined by the organ in which the cell operates) ceases because of both decreased energy and the accumulation of toxic by-products of anaerobic metabolism. The cell soon becomes unable to perform its necessary homeostatic processes. Intracellular structures called lysosomes (bags of toxic enzymes) rupture and digest the cellular contents. The cell membrane becomes incompetent, and with increasing time, ruptures. This releases the toxic intracellular enzymes and metabolic by-products into the circulation. The delivery of these toxic substances to other cells results in further cellular dysfunction and damage. Tissues and subsequently organs, can no longer function. The organism finally dies if the process is not reversed (irreversible shock).

The goal of resuscitating the patient in shock is to improve cellular perfusion and oxygenation.

CLASSIFICATIONS OF SHOCK

Not all shock is caused by hypovolemia. This is important not only for the purpose of diagnosis, but also for management and resuscitation.

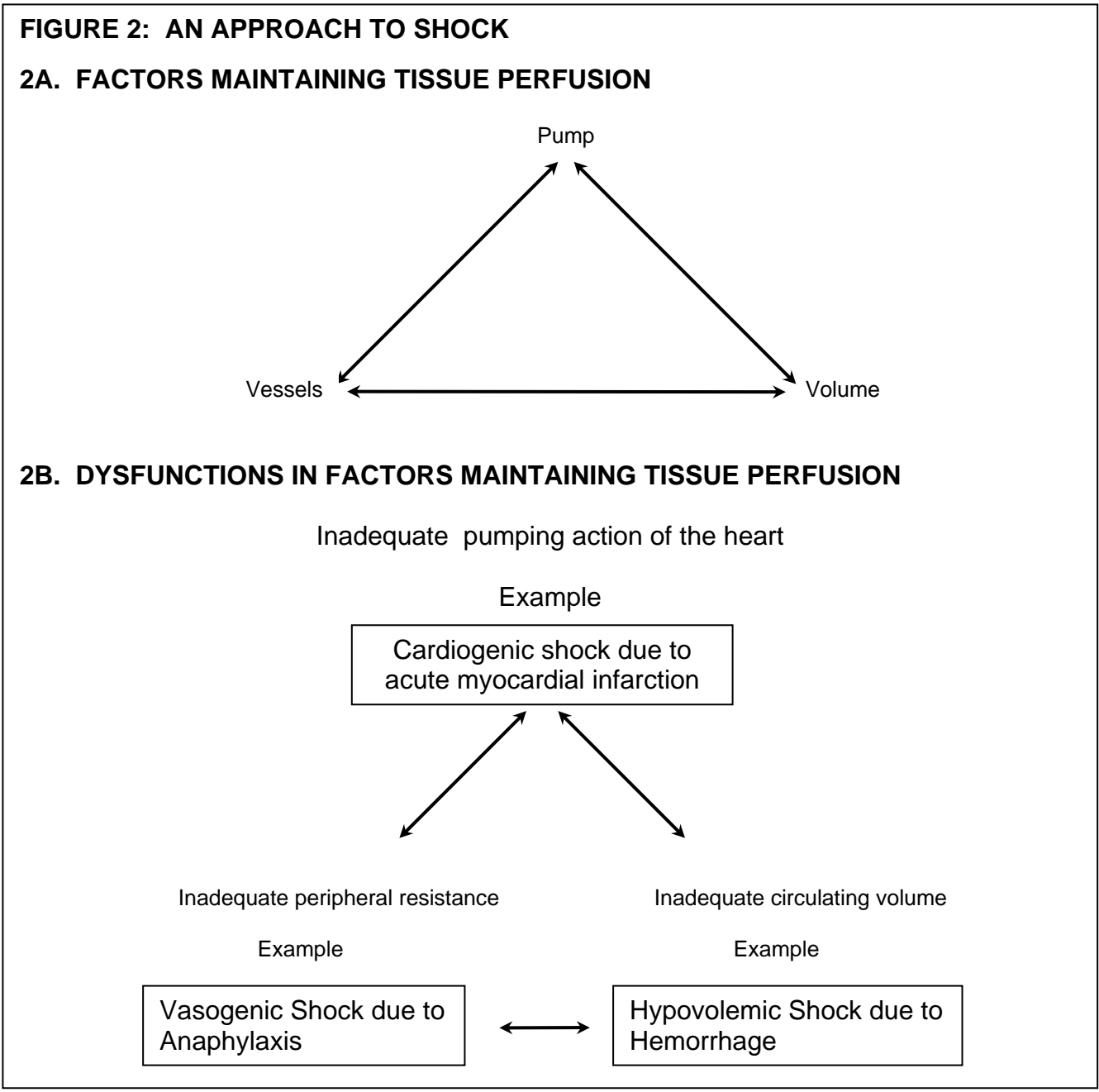
The individual who is in shock because of a tension pneumothorax, will not respond to crystalloid resuscitation (volume infusions of I.V. fluids such as normal saline). An individual who is in anaphylactic shock, will require the administration of epinephrine, to control the process of mediator release and subsequent vasodilation and capillary leak. A simple classification of the causes of shock is listed in Table 1.

TYPES	CAUSES
HYPOVOLEMIC Hemorrhagic Non-Hemorrhagic	<ul style="list-style-type: none"> o Internal loss, e.g. GI bleed o External loss, e.g. compound fracture o GI losses, e.g. prolonged severe diarrhea in infants o Renal losses, e.g. excessive use of diuretics o Cutaneous losses, e.g. heat exhaustion, burns.
MECHANICAL/ OBSTRUCTIVE (Mechanical interference with blood flow)	<ul style="list-style-type: none"> o Tension pneumothorax o Cardiac tamponade o Massive pulmonary embolus o Dissecting aortic aneurysm
CARDIOGENIC (Impaired function of the heart as a pump)	<ul style="list-style-type: none"> o Myocardial infarction/contusion o Arrhythmia
NEUROGENIC (Block of the sympathetic out flow resulting in peripheral vasodilation)	<ul style="list-style-type: none"> o Spinal shock o Severe head injury or intracranial vascular event o Overdose
SEPTIC AND ANAPHYLACTIC	<ul style="list-style-type: none"> o Vasodilation caused by humoral or toxic substances acting on blood vessels.
OTHERS (Rare)	<ul style="list-style-type: none"> o Addison's disease (adrenal gland failure) o Myxedema coma (thyroid gland failure)

A simple mnemonic which stresses the important causes of shock is outlined below:

- S** Septic, Spinal (Neurogenic)
- H** Hypovolemic (+/- Hemorrhagic)
- O** Obstructive (Mechanical)
- C** Cardiogenic
- K** Anaphylactic “k”

A similar and acceptable approach is to think of shock as secondary to dysfunction of one of three major factors.



TYPES OF SHOCK

MECHANICAL/ OBSTRUCTIVE SHOCK

It is important to note that the mechanical obstruction of blood flow may result in shock. A trauma patient with a tension pneumothorax must be diagnosed rapidly as therapy is specific (release of air from within the pleural space) and dramatic.

With a tension pneumothorax, the raised intrathoracic pressure impedes venous return, and the shift of the mediastinum may contribute to impaired cardiac function. A massive pulmonary embolus impedes blood flow to the lungs and subsequently to the left ventricle. Cardiac tamponade results in impaired diastolic filling since fluid/blood in the pericardial sac compresses the heart. A dissecting aortic aneurysm can obstruct blood flow distal to the left ventricle and result in widespread tissue hypoperfusion and hypoxia.

An important physical finding with both mechanical shock and cardiogenic shock is distended neck veins indicating raised central venous pressure. Raised central venous pressure rules out hypovolemia as the cause of the shock state (at least in isolation) and should raise suspicion of a mechanical or cardiac etiology.



Clinical vignette

You may recall that the pressure in the vena cava is 0-8 mmHg. Therefore it's easy to see how a mediastinal shift with a tension pneumothorax might compress the vena cava reducing blood return to the heart (preload).

SEPTIC SHOCK

Septic shock is precipitated by the release of endotoxins by microorganisms (usually gram negative bacteria) into the bloodstream. This results in decreased vascular resistance, peripheral pooling of blood and ultimately capillary leak with fluid extravasation. In sepsis, the effective blood volume is low relative to the size of the "tank" and the end result is a decreased blood supply to major organs and ultimately organ failure. Most commonly seen in the elderly with underlying medical illnesses such as diabetes or cancer as well as in the very young. The signs and symptoms are variable in the early and last stages of sepsis as outlined in Table 2. Clinical management is based on use of inotropes (drugs that increase cardiac contractility) or vasopressors and/or fluid volume resuscitation with normal saline or Ringers Lactate. Hospital or critical care treatment would include the use of antibiotics, steroids and general supportive measures. Mortality is approximately 45%¹.



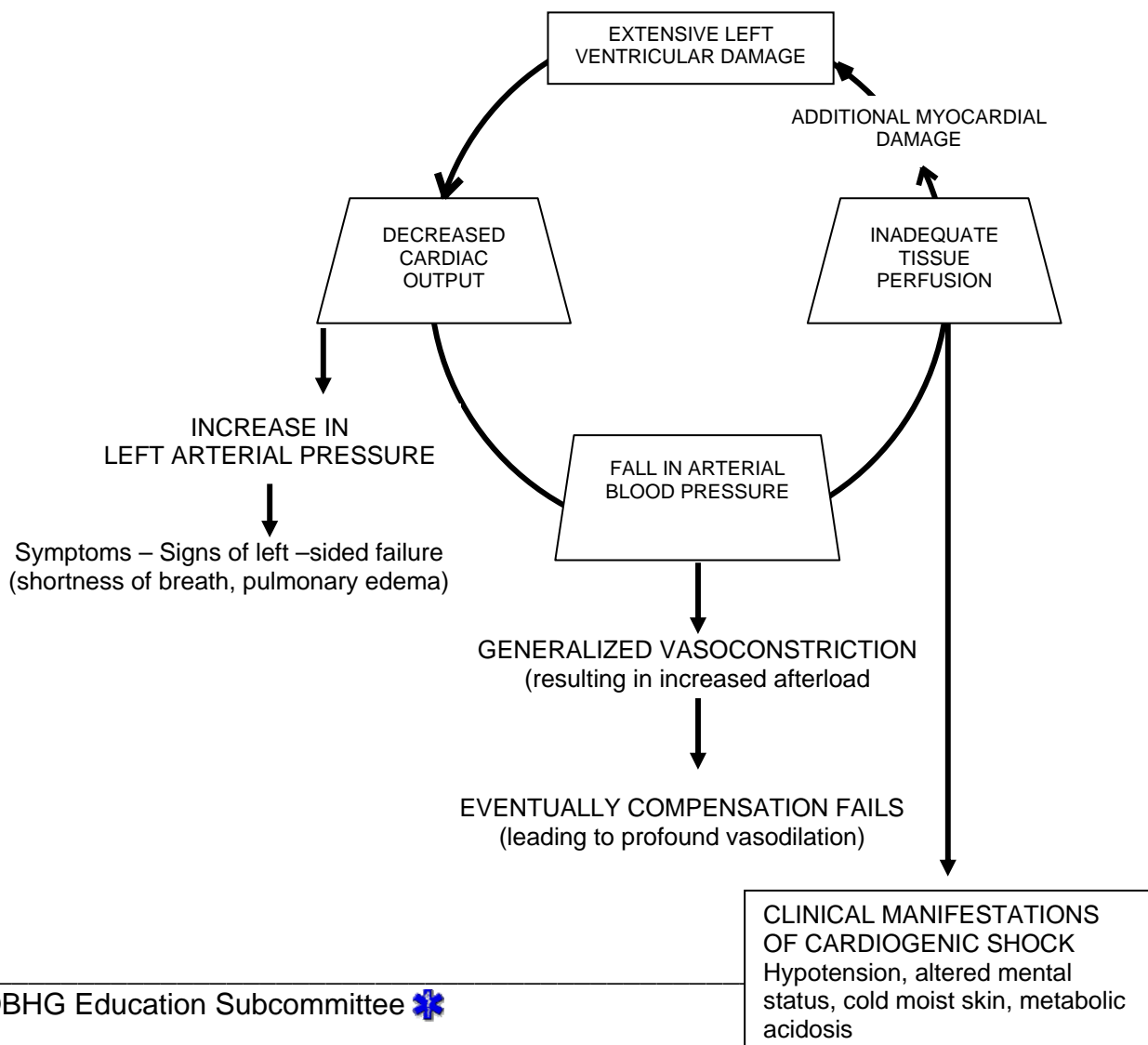
Clinical vignette

Suspect septic shock with the following: Recent known infection, urinary catheterization, pneumonia, infected surgical wound, fever or unexplained hypothermia, hypotension (<90 mmHg systolic) that may not respond to fluid resuscitation, tachycardia, tachypnea.

CARDIOGENIC SHOCK

Cardiogenic shock is the most severe manifestation of decreased left ventricular pumping function. Cardiogenic shock develops because the left ventricle is unable to maintain the cardiac output at a level necessary for adequate tissue perfusion. The majority of patients with cardiogenic shock will have had a massive left ventricular infarction. Other potential causes are a right ventricular contusion (occurring with deceleration injuries) in a trauma patient resulting in damage to one of the AV valves, or a dysrhythmia (e.g. unstable tachydysrhythmias such as ventricular tachycardia, atrial fibrillation, etc).

FIGURE 3: CARDIOGENIC SHOCK IN THE SETTING OF MYOCARDIAL INFARCTION



In the setting of acute myocardial infarction (AMI), most patients who die of cardiogenic shock usually have more than 40% of the myocardium destroyed (not necessarily from the initial infarction). The infarcted area continues to enlarge during the course of cardiogenic shock and since blood flow through the coronary arteries decreases during shock, the myocardium is further deprived of oxygen. This further impairs myocardial contractility of the uninjured ventricular segment and at the same time promotes additional tissue destruction. A vicious cycle is set up which explains the high mortality (>80%) from cardiogenic shock in the setting of AMI.

ACUTE RIGHT VENTRICULAR INFARCTION - SHOCK

Right ventricular (RV) infarction occurs in approximately 25-30% of patients who experience an acute inferior left ventricular infarction. Most often this results from a proximal occlusion of the right coronary artery (RCA) which feeds the SA node, AV node, right ventricle and the inferior left ventricle in 90% of the population. Consequently, the right ventricle is not able to contract effectively and the result is hypotension and cardiogenic shock. However, unlike the cardiogenic shock that most of us envision, the clinical presentation of RV infarction has all of the typical elements of shock such as hypotension, altered mental status, shortness of breath, but pulmonary edema is absent, the neck veins are generally distended and tachycardia may be absent, and in fact the patient may be bradycardic. This occurs because the SA node may be ischemic or excess vagal tone may blunt the reflex tachycardia typically seen in shock.



Clinical vignette

Acute Right Ventricular Infarct: Signs & symptoms consistent with cardiac ischemia/ AMI, hypotension, bradycardia or normal heart rate, SOB, **clear chest**, distended neck veins.

12 Lead ECG: look for ST elevation in the inferior Leads (II, III, aVF) and ST elevation in RV4.

Note: Primary Care Paramedics are encouraged to learn 12 Lead ECG interpretation as ECG changes may be seen in the prehospital setting that may be of value for hospital staff.

* Adapted from Denny, M.P.: Septic Shock, JEN 3:19, Jan-Feb 1977.

** Adapted from "Therapeutic Considerations in Critical Care Medicine, Hemodynamic and Respiratory Aspects of Shock", Kalamazoo, Michigan, 1976, The Upjohn Co.

ANAPHYLACTIC SHOCK

Anaphylaxis is an acute, severe, systemic allergic reaction caused by the release of chemical mediators (histamine, prostaglandins, leukotrienes and kinin) after an interaction with IgE antibodies on the surface of mast cells and basophils. These chemical mediators result in widespread vasodilation (predominant cause of shock), capillary leak with decreased blood volume and tissue swelling, bronchospasm, increased mucus production and shock. The most common causative agents are medications (especially after parenteral administration), foods and insect stings/bites. Simple allergic reactions may take minutes to hours, however anaphylaxis typically occurs within minutes. A high index of suspicion is therefore necessary and careful history may reveal an allergy history.

Anaphylaxis can cause significant organ compromise and death within minutes.

The primary systems involved in anaphylactic shock are:

1. Respiratory
 - o Upper airway obstruction secondary to edema
 - o Bronchospasm secondary to bronchoconstriction.
2. Integumentary
 - o Urticaria (hives)
 - o Local swelling
3. Gastrointestinal
 - o Nausea, vomiting and diarrhea secondary to chemical mediator release and sympathetic nervous system stimulation.
4. Circulatory
 - o Widespread vasodilation leading to hypotension resulting in circulatory collapse.

The mainstay of treatment for anaphylactic shock is the administration of epinephrine to counteract the widespread vasodilation, reverse the mediator response and decrease airway swelling and bronchospasm. Fluid resuscitation and the use of antihistamines and steroids may also be appropriate.

Note: Further treatment with salbutamol for wheezing may be appropriate if bronchospasm does not respond adequately to epinephrine.

HEMORRHAGIC SHOCK

As emphasized previously, not all shock is hemorrhagic. A frequent mistake is to assume that the trauma patient is in shock from hypovolemia (as opposed to a tension pneumothorax) or

that an elderly confused, alcoholic, male is in shock from a GI bleed (as opposed to septic shock). It is important to rule out other potential causes for shock which may have specific treatment. The module on hypovolemia and its management will focus on this problem.

Conversely, if a patient with a severe head injury is in shock, it is much more likely due to hypovolemia than the head injury and should be treated as such.

Table 3 is useful in that it correlates physical findings with the approximate blood loss. Calculations are based on a 70 kg male with a blood volume of 70 mL/kg. The physical findings may overlap between different classes of shock, and the principles are more important than specific numbers.

TABLE 3 SEVERITY OF HEMORRHAGIC SHOCK*				
	CLASS I	CLASS II	CLASS III	CLASS IV
Blood loss in mL	Up to 750 mL	1000-1250 mL	1500-1800 mL	2000-2500 mL
Blood loss in %	Up to 15%	20-25%	30-35%	40-50%
Pulse rate	72-84	>100	>120	140 or greater
Blood pressure	118/82	110/80	79-90/50-60	<50-60 systolic
Capillary blanch test	Normal	Prolonged	Prolonged	Prolonged
Respiratory Rate	14-20	20-30	30-40	<35
CNS – mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused, lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid + blood	Crystalloid + blood

(use 3:1 rule for fluid resuscitation)



Clinical vignette

OXIMETRY: Remember that the SpO₂ in a hypovolemic patient may be normal. However, because of blood loss (loss of hemoglobin), the body's oxygen carrying capacity will be reduced. Therefore, supplemental oxygen is critical in the setting of hypovolemia to increase the amount of dissolved oxygen in blood plasma.

* Adapted from the American College of Surgeons. Classification of Hemorrhagic Shock, Advanced Trauma Life Support Course.

COMPENSATORY MECHANISMS IN SHOCK

DEFINITION

When a patient is in shock, the body will attempt to maintain an adequate blood flow to **vital organs** (brain, heart, kidneys), to maintain cardiac output and tissue perfusion.

There are three major mechanisms employed to achieve this:

- o Nervous
- o Chemical
- o Hormonal

If vital organ perfusion is adequate the shock is said to be **compensated**. This is, however, an unstable hemodynamic situation which can deteriorate with time or additional stress on the system, **i.e.** further blood loss, myocardial dysfunction secondary to prolonged ischemia.

COMPENSATORY MECHANISMS EMPLOYED

1. **Neurogenic compensation** is the most rapid and is known as the fight or flight response. It consists primarily of arterial and venous vasoconstriction (in an attempt to maintain an adequate perfusion pressure) and an increased heart rate (in an attempt to maintain cardiac output). This means that there is preferential perfusion of the brain and heart.
2. **Chemical compensation** occurs within thirty minutes. The decreased cardiac output and increased oxygen extraction by the tissues leads to a decreased arterial PaO₂ causing the chemoreceptors in the aorta and carotid arteries to stimulate the respiratory centre. The respiratory centre responds with a respiratory alkalosis. Unfortunately this leads to vasoconstriction of cerebral vessels, cerebral ischemia and changes in level of consciousness.
3. **Hormonal compensation** can occur when impulses arrive via the sympathetic nervous system. Three major types of hormonal compensation can result:
 - a) The adrenal medulla is stimulated to release its hormones (epinephrine/norepinephrine – potent vasoconstrictors).
 - b) Decreased blood flow to the kidneys leads to the activation of the **Renin-Angiotensin system**. Renin converts angiotensinogen (protein) into angiotensin I. Increased angiotensin I passes through the lungs where an enzyme called angiotensin converting enzyme (ACE) converts angiotensin I to angiotensin II. The hormone angiotensin II is a powerful vasoconstrictor and it also stimulates the adrenal cortex to release aldosterone. Aldosterone causes the kidneys to

- retain sodium . The retained sodium results in an increased intravascular volume, thereby increasing systemic perfusion.
- c) The hypothalamus stimulates the anterior pituitary to secrete adrenocorticotrophic hormone (ACTH) which in turn stimulates the release of adrenal cortical hormones (glucocorticoids). These hormones influence the metabolism of carbohydrates, proteins and fats, and decrease the permeability of capillary walls. This helps to limit the loss of intravascular fluid.

It should be noted that these compensatory mechanisms are time-limited, and although attempting to improve **vital organ perfusion**, result in a further **widespread** reduction in tissue perfusion secondary to generalized vasoconstriction. These compensatory mechanisms are less efficient in infants and toddlers, with advancing age or with underlying concurrent illnesses.

SUMMARY

As noted in the discussion above, shock may be considered as a spectrum from early reversible shock to late irreversible shock. Each patient must be considered as an individual and the ultimate outcome will depend on the past health, age, pre-existing illnesses, etc.

Assessment of the patient will focus not only on diagnosing the presence of shock but also on elucidating the cause(s) so that appropriate therapeutic interventions may be undertaken. As noted above, certain specific treatments, **e.g.** pericardiocentesis, may only be carried out in a hospital setting and rapid transport may be the most appropriate prehospital management.

The principles of assessment, and management (oxygenation and management of airway, hypovolemia, selected emergencies) will be covered in other modules.

The major principles of resuscitation for the patient in shock are to:

1. Improve cellular perfusion
2. Improve cellular oxygenation.

REFERENCES

1. J. Stephan Strapczynski: **Shock, Septic.** eMedicine .com Journal, July, 2002:
<http://www.emedicine.com/EMERG/topic533.htm>

**ADVANCED LIFE SUPPORT
PRECOURSE
SHOCK**

SELF-ASSESSMENT

Marks

[2] 1. a) The clinical aim of shock therapy is to restore two processes at the cellular level. What are they?

[2] b) What is the relationship between these processes?

[4½] 2. For each of the three etiologies of shock shown below (Pump, Vessels, Volume), list three possible causes for shock. (1/2 mark for each correct answer).

PUMP:

VESSELS:

VOLUME:

[3] 3. The cell prefers to utilize a) _____ and b) _____ to make c) _____.

Marks

[3] 4. Briefly explain the etiology of the metabolic acidosis seen with shock.

[3] 5. Explain the statement “hypotension and shock are NOT synonymous”.

[1] 6. What is the approximate total blood volume of a healthy 100 kg man?

[1] 7. Slight tachycardia and mild anxiety may be your only clues to early detection of an occult bleed. Why?

[4] 8. a) Which four primary body systems are affected in anaphylactic shock?

b) Name one clinical manifestation for each system affected.

Marks

[1] 9. a) One physical finding will rule out hypovolemia as a cause of shock. This is _____.

[2] b) This finding is associated with _____ or _____ causes of shock.

[1] 10. Clinical findings particular to cardiogenic shock are:

[4] 11. The patient's ability to compensate for shock is influenced by _____, _____, _____, and _____.

31 ½ TOTAL

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SELF-ASSESSMENT ANSWERS

1. a) Oxygenation and perfusion.
b) Oxygenation is dependent upon delivery of oxygen to the blood via the respiratory system and the ability of the hemoglobin to transport it to the cell.
2. Pump: a) M.I.
b) Myocardial contusion
c) Dysrhythmia

Vessels: a) Sepsis
b) C-spine injury
c) Anaphylaxis

Volume: a) GI losses
b) Renal losses
c) Hemorrhage
3. a) O₂
b) Glucose
c) ATP or energy
4. Subtract one mark for each key concept missed to zero.

Decreased oxygen delivery to cells results in **metabolism without oxygen: glycolysis** leads to **pyruvate** production, which is metabolized to **lactic acid**, creating metabolic acidosis.
5. What is of note is whether the total clinical picture of the patient indicates that his cells are oxygenated and perfused.
6. 7 litres (7,000 mL)
7. Neurogenic (SNS) compensation or the “fight or flight” response is responsible for the vasoconstriction, tachycardia and anxiety.

8. (1/2 mark for each correct answer)

- | | | | |
|----|--|----|---|
| a) | <input type="radio"/> Respiratory | b) | <input type="radio"/> Bronchospasm or edema |
| | <input type="radio"/> Integumentary | | <input type="radio"/> Hives |
| | <input type="radio"/> Gastrointestinal | | <input type="radio"/> Nausea, vomiting, or diarrhea |
| | <input type="radio"/> Circulatory | | <input type="radio"/> Hypotension |

10. a) Jugular venous distension OR distended neck veins.

b) Cardiogenic or mechanical (obstructive).

11. (1/2 mark for each correct answer)

Pulmonary edema, JVD

12. Duration of shock
Severity of shock
Age
Concurrent illness

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EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

Reading _____ hours
Self assessment _____ hours
Total time _____ hours

2. Were the objectives of the module clearly stated?

yes no
If no, please comment.

3. Did you see any of the resource materials?

yes no
If yes, which items

Were they helpful?

4. Were the reference notes adequate?

yes no
If no, please comment.

5. Were the reference notes easy to follow?

yes no

If no, please comment.

6. Were the examples provided satisfactory?

yes no

If no, please comment.

7. Were any of the self-assessment questions poorly worded?

yes no

If yes, please specify.

8. Was the level of the module satisfactory for your program of study?

yes no

If no, please comment.

Base Hospital _____

9. General comments or suggested improvements.